hours is a normal post-operative condition, provided it be not sustained.

2. Probably the maximum leucocytosis in the majority of cases occurs within the first twelve hours after operation, and is very transient.

3. The leucocytosis in the normal reparative process bears but slight relation to the pulse and temperature.

4. A post-operative leucocytosis of 10,000 or more *above the individual normal* sustained for more than a few hours may be looked upon with suspicion.

5. The apparent increase in number of erythrocytes following operation is not caused by an actual increase of red cells in the circulating blood.

To Drs. Eugene Boise and Richard R. Smith, of Butterworth Hospital, and Drs. Schuyler Graves and O. E. Herrick, of the Union Benevolent Association Hospital, I am indebted for the opportunity of making these observations, every courtesy possible being extended me by all of these gentlemen during their respective services.

ENTRANCE OF AIR INTO THE VEINS, AND ITS TREATMENT.

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Although the dangers accompanying sudden entrance of air into the venous circulation were recognized early in the eighteenth century by such observers as Redi, Valsalva, and Morgagni, it remained for Beauchesne, in 1818, to first report a death, verified by autopsy, due to entrance of air into the jugular vein during an operation for the removal of a tumor from the neck.

The discussion following Beauchesne's report has been carried on during the past century by such clinicians and experimentalists as Barlow, Mirault, Delaporte, Ammussat, Cormack, Wattman, and others—during the early part of the nineteenth century—and later by Senn, Hare, Treves, Porter, Hektoen, Draper, and Davidson.

The conclusions which have been reached are various. On the one hand, we have heard warnings against its dangers; others have endeavored to dispel the dread of this accident existing among surgeons. Some have taught that the merest bubble of air entering the veins may cause alarming symptoms, possibly death; while others have claimed that large quantities of air may be injected into the veins near the heart with no ill effects save a slight, temporary fall in the blood-pressure. Even among those who have believed in the dangers attending the accident, opinions have differed as to the direct cause of death.

Treves believed that death was due to arrest of pulmonary circulation, the air in the right heart being mixed with blood, and preventing action of the tricuspid and pulmonary valves, but little blood reaching the left ventricle, and anæmia of the brain following.

Cormack thought that death was due to gaseous distention of the right heart.

Bell asserted that the cause of death lay in the transference of air to the base of the brain, with consequent respiratory paralysis.

Virchow claimed that complete air embolus of the pulmonary artery, with consequent loss of blood to the coronary circulation and arrest of the heart in diastole, was the immediate cause of death.

In citing these cases I have by no means exhausted the differences in opinion which have existed during the past century among eminent observers.

While the cause of death has been a subject of much controversy, the fact that death does occur and is due directly to the entrance of air into the veins is acknowledged by surgeons generally, there being few notable exceptions.

Hare, in 1889, published a paper in the *Therapeutic Gazette* voicing views on this subject at variance with the popular idea. In comparing his paper with that of Senn's, published four years previously, we must be impressed with the fact that in experimental work personal equation plays no insignificant part. Both are men whose opinions carry weight; and yet we find, on the one hand, Hare, after experimenting with some seventy dogs, asserting that even large quantities of air do little harm, while small amounts, varying from 20 c.c. to 40 c.c., injected into the jugular vein cause little more than slight transitory fall in blood-pressure; on the other hand, Senn found that small quantities of air when injected into the jugular vein proved fatal in a large percentage of his dogs.

While this accident occurs more commonly during operations in the neck and axillary regions, the veins in other parts of the body are by no means exempt.

Davidson reports the case of a Hindoo woman who died suddenly, after a normal labor, and in whom the autopsy revealed dilated uterine veins, congested lungs, and a right ventricle filled with mixed air and blood.

So, also, Wyman reports a death following rapidly the aspiration of a pelvic tumor, where the autopsy showed a right heart dilated with air, while neither ventricle contained any blood.

Volkmann reported a death due to entrance of air while aspirating a longitudinal sinus.

Bergmann describes the case of a man whose occiput was injured; the man died in four hours. On post-mortem examination both lungs were found œdematous, and there was a very extensive form of air embolism, the air probably entering through the injured torcular.

A death has been reported from entrance of air into the sinuses of the dura mater. However, so much more frequently has this accident occurred during operations in the region of the axilla and neck that this has come to be known as the "danger zone."

Senn says: "Clinical observation and experimental research have established the fact that the venous circulation is directly influenced by respiration within a certain area, and that inspiration of air, in the majority of cases, takes place in the veins thus affected, constituting the justly much-dreaded 'danger zone.'"

It is in these veins near the heart that the so-called suction force is exerted. When a vein is completely severed and its lumen closed by collapse of its walls the danger is practically *nil*. Partial division or nicking of the vessel constitutes the condition favorable to the drawing of air into the vein.

The peculiar relation of the external jugular vein to the cervical fascia and of the axillary vein to the costo-coracoid fascia renders the lumen of these vessels patulous even when cut through. This is also true of veins whose walls have been diseased, thickened by calcareous deposits, or in such conditions as periphlebitis, where the surrounding inflammatory tissue is intimately connected with the walls of the veins. Thus the factors in this region upon which this accident depends are two—patulous veins and suction force exerted during inspiration.

The accident has occurred with greatest frequency during removal of tumors from the neck and axilla, amputation at the shoulder-joint, resection of the scapula, ligation of the subclavian, and venesection of the jugular vein; but it may occur in the most trivial operation in this region, and more frequently in the cervical veins than in the axillary.

The symptoms attending this accident as described by various surgeons and coinciding with my own observations in experiments on dogs are: A hissing noise accompanies the entrance of air into the vein; the blood-pressure immediately falls; the heart—at first tumultuous, with a churning sound plainly heard over the præcordial area, synchronous with the heart action—becomes rapidly weak and irregular; convulsive twitchings of the entire body occur; at this time (and the preceding symptoms have occupied scarcely thirty seconds) the pulse has disappeared, the heart has ceased except for an occasional impulse, the breathing becomes labored and rapid, and after a few violent inspiratory efforts the animal dies. The picture is one of cardiac failure, with secondary respiratory paralysis due to anæmia of the brain centres.

While working with Dr. Kemp in the Columbia Physiological Laboratory, two years ago, to determine the effects on blood-pressure of infusion with normal salt solution introduced at temperatures ranging from 105° to 120° F. the following accident occurred:

A moderate-sized dog was given a dgm. of morphine at 10 A.M.; at 10.45 A.M. ether was administered. When the animal was completely anæsthetized a tracheotomy was performed for convenience in administering the ether during the further course of the experiment.

The right carotid was then opened, and into it was inserted a manometer tube, which in turn was attached to the kymograph, so that any changes in blood-pressure occurring during the experiment might readily be noted.

The right femoral artery was exposed, and into it a canula was tied. As a final procedure the femoral vein was opened, and through the canula placed in its lumen we expected to perform our infusion. The saline infusion was then prepared at a temperature of 105° F. and placed in the douche bag. The kymograph was set in motion and the normal blood-pressure noted—*i. e.*, 182 mm. We now removed through the canula placed in the femoral artery 100 c.c. of blood; the pressure slowly fell during the bleeding to 110 mm. After waiting two minutes for spontaneous recovery in pressure, which invariably occurs after bleeding, we prepared to start our infusion.

The canula in the femoral vein was carefully filled with salt solution, so that no air might enter when the infusion was started. Through some misunderstanding the same precaution was not exercised on the tube of the douche bag, consequently it was filled with air between the clip and the distal end; so when it was attached to the canula in the vein and the clip opened, a column of air 3 feet long and 6 mm. in diameter was forced into the vein by the pressure of the fluid in the bag; about 12 c.c. of air must have been injected in this way. The air was plainly seen rushing through the glass canula. The blood-pressure fell in a few seconds to near zero. (See Fig. 1.) The churning sound observed in these cases was distinctly heard; the heart's action became at first tumultuous, and then rapid and weak; convulsive muscular twitching began; respirations were labored and inspirations deep and gasping; death seemed certain. Instead of removing the canula and stopping the infusion the saline was immediately increased in temperature to 120° F., and artificial respiration by means of Hoyt's artificial respiration apparatus was begun. Slowly the action of the heart returned; a slight tracing reappeared on the drum, and the blood-pressure slowly arose. The infusion was continued, being given very slowly; artificial respiration was fixed at 20 per minute; in fifteen minutes the heart had regained its expansile force, the tracing on the drum was normal, and respiration was much improved. Accordingly both infusion and artificial respiration were stopped. The blood-pressure remained at 156 mm., and we continued our original intentions on the same dog for the subsequent hour and a half.

This experiment was particularly valuable because of the fact that the entrance of air was entirely accidental, and happened under conditions that might well have occurred clinically. It seemed to show that the entrance of air into a vein under pressure was dangerous and might lead to a fatal termination. It also seemed that in the treatment of this condition direct venous infusion with normal salt solution at a temperature of 115° to 120° F. and artificial respiration were important factors. I was lead by this result to continue the experiments on a series of dogs at the Columbia Physiological Laboratory last winter.

In the first five experiments the following routine was followed: The dog was given morphine in dosage from 5 cgm. to 1 dgm., according to its size; after waiting half an hour the animal was etherized and tracheotomy was performed. The femoral vein was exposed for injection of air and infusion; a canula with a stopcock attachment connecting with the atmospheric air, in order that the syringe might be filled without removal from the vein, was inserted and tied in place, and the carotid artery on the right side was then exposed and connected by means of a manometer tube to the kymograph. I have chosen Experiment 3 as a fair representative of this class.

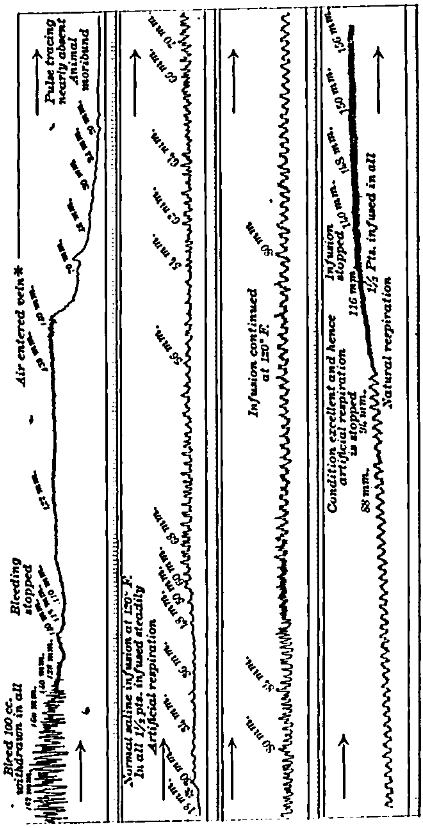
A white setter, weighing forty pounds, was given 1 dgm. of morphine, and 75 c.c. of air was injected into the vein. The pressure dropped from 124 mm. to 8 mm. in a period of time covering fifty seconds. Immediately following the entrance of air the heart became tumultuous; a churning sound was plainly heard in the chest; in a few seconds the heart became weak and irregular, the femoral pulse disappeared, and finally the heart ceased to beat perceptibly. The respirations were not affected until some seconds after the heart began to show the first changes; then they became labored and more rapid. This was followed by the peculiar forced inspirations noted in all these cases, and the respirations did not cease entirely until one minute after the last heart tracing was noted on the drum. An infusion of normal salt solution at a temperature of 120° F. was given, without the slightest result; the dog was dead. The autopsy revealed the right ventricle distended with air; the large venous trunks contained many air bubbles, as did the pulmonary artery. The coronary vessels were found filled with air emboli and the heart was fibrillating; this continued for about fifteen minutes.

In all these dogs the air was injected with considerable force; the maximum amount used in any individual case was 75 c.c. Infusion was resorted to in six dogs, with recovery in two. In one of the dogs which recovered I believe such would have been the case with no treatment; thus there was but one favorable result following treatment by infusion alone. (See Fig. 1—accidental entrance of air during infusion.)

In one dog, after a temporary rise in blood-pressure, too much infusion was allowed to enter the vein, and it died of pulmonary œdema.

In another dog death occurred when artificial respiration was begun after a temporary rise in blood-pressure had been noted following infusion.

FIG. 1.



On the rest of the dogs experimented upon infusion had no effect whatever. In all the primary effect of air injection was on the heart, the respiratory symptoms being secondary. An autopsy was performed after each experiment, except Experiment 1, where entrance of air was accidental, and in all the animals the coronary vessels were found full of air emboli and the heart fibrillating; in four of the five the right ventricle was distended with air.

I am inclined to believe that death in these cases was due to one of two possible causes: First, gaseous distention of the right ventricle, with loss of contractile power, failure of both pulmonary and tricuspid valves to act, failure of blood to reach the left ventricle, and consequently anæmia of the brain centres. The second, and it seems to me the more important factor in the death of these animals, was the plugging of the coronary vessels with air emboli. The heart being deprived of its circulation was thrown into fibrillation, and death ensued. This was observed by Cunningham in 1898. Erichsen, Cohnheim, Rechberg, Porter, and others found that by tying off the coronary vessels or injecting them with a preparation of wax, paraffin, and soot, thus completely shutting off the intrinsic circulation of the heart, the heart becomes irregular, the pressure sinks, and the ventricles beat arythmically; that is, they cease to beat as a unit, but individual muscle fibres continue an inco-ordinate contraction for some time. This is precisely the condition observed in these dogs.

I cannot agree that respiratory failure plays a primary part in the result, as is believed by Hare and others. Hare reports two cases in which he injected the carotid artery; in one 40 c.c. of air was used, and in the other 20 c.c.; each was equally fatal, and respiratory failure was in these cases the primary cause of death, the heart beating some time after respiration ceased. His deductions based on these experiments are as follows:

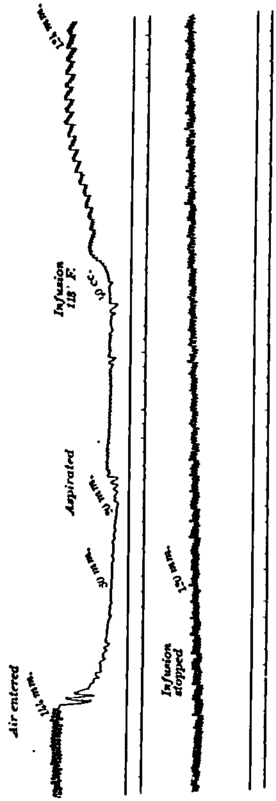
"Under such circumstances" (air injected into the carotid) "the results are quite different from those seen after opening the jugular vein, but nevertheless totally disprove the theory that it is by cardiac failure that death under such circumstances occurs." This statement holds good only in so far as injection of the arteries with air is concerned, air emboli being carried directly to the base of the brain and the respiratory centres, while, when the venous system is injected the air is carried to the right ventricle, and the resulting mechanical dilatation of that receptacle causes anæmia of the brain centres and secondary respiratory paralysis. If Hare means to apply the results of his experiments with injection of air into the arteries to similar injections of the veins I think his conclusions are obviously erroneous. (See Fig. 5, Experiment 17.) Rapid injection of air was an important factor in obtaining the preceding results, for, as will be seen later, considerable quantities of air may be slowly injected without serious effect.

I was impressed with two facts in my first experiments: First, that entrance of air into the veins is a serious accident, which may well result fatally; and secondly, that when the right heart is distended with air, infusion can be of little service until that air is removed. I believe with Treves that artificial respiration is unnecessary, if not contraindicated, there being no primary respiratory failure, and the dyspnœa being due not to lack of air, but lack of blood in the lungs.

In 1885 Senn, after injecting air into the veins of dogs, aspirated the right heart by means of a needle about 2 mm. in diameter, forced through the chest wall into the ventricle; 30 per cent. of his dogs recovered after this treatment. It seemed to me that if this were followed by infusion of saline at high temperatures we might expect even a higher percentage of recoveries. Accordingly I experimented on some eleven dogs, with this object in view. The same routine was followed as in the former experiments, with two differences: the external jugular vein was used for our injections of air instead of the femoral, and in the treatment an aspirating needle, 2 mm. in diameter, was forced into the right ventricle, and the air removed by means of Hoyt's vacuum pump before infusion was begun. I shall let Experiments 10, 13, 15, and 17 represent this class.

Experiment 10 (No. 1).—This dog—a heavily built bulldog, weighing 34.5 pounds—was given 1 dgm. of morphine about an hour before the anæsthetic was commenced. As we intended to allow this dog to come out of ether we did not do tracheotomy. As much care as possible was used in asepsis; after anæsthetizing, the carotid and jugular were exposed and treated as in the other experiments. Everything being ready, the kymograph was set in motion and the normal pressure noted; in this case it was 148 mm. After waiting five minutes 75 c.c. of air from an Ultzman syringe was injected into the left jugular vein. The effect was instantaneous; within ten seconds the pressure fell from 140 mm. to 40 mm.; the heart was tumultuous; the churning sound before described was plainly heard; in a few more seconds the pressure had fallen to 18 mm. (which is practically zero, as it will never fall to actual zero, even when the animal is quite dead, until the arterial canula is removed); the heart grew rapidly weak, until all pulse tracings finally disappeared from the drum; the respirations still continued, however, though labored and gasping at this time. We then inserted a needle into the fourth left interspace, about an inch from the sternum; the vacuum apparatus was set in motion and a vacuum created in the bottle; the needle was then thrust obliquely into the right ventricle, and about 150 c.c. of air mixed with blood was drawn off. The pressure arose immediately, the pulse tracings reappeared on the drum, and the cardiac expansions increased; at this point infusion was begun, and about thirteen ounces of normal salt solution at a temperature of 120° F. was allowed to run in very slowly. One minute after this treatment the pressure had risen to 128 mm.; infusion was stopped, the vessels were tied, and the wound was closed with catgut sutures. The dog was then placed in a cage, was wrapped in blankets, and was surrounded by hot-water bottles. The ether was stopped at 1 P.M.; at 2 P.M. the dog came out from under the influence of his anæsthetic; at 5 P.M. the same day he was weak, but able to stand. At this time half a pint of milk was given him, which he retained. The next day he got "regular diet." During the next five days he continued to do well, took his nourishment eagerly, and, with the exception of a stitch abscess in the femoral incision, was apparently as well as before the operation.

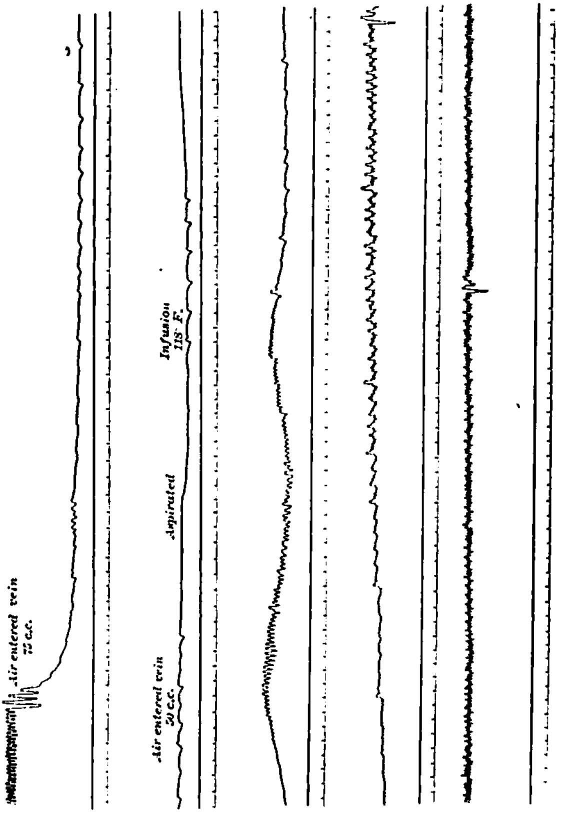
FIG. 2.



No. 2.—On the morning of the sixth day the same dog was again etherized, the opposite jugular was exposed and opened, and 75 c.c. of air was injected. Immediately the same symptoms developed as were present in this dog six days previously. No treatment was resorted to in this case, and the dog died. After waiting for five minutes the chest was

opened, the right ventricle was found distended with air, the auricles were spasmodically contracting, the coronary vessels were filled with air emboli, and the heart was fibrillating; this continued for fifteen minutes. The old puncture scar was visible at the base of the right ventricle; there was no blood in the pericardium.

FIG. 8.



Experiment 13.—A dog weighing forty-four pounds was given 8 cgm. of morphine, anæsthetized with ether, and dealt with in the manner above described. Everything being ready, the kymograph was set in motion at 11.38 A.M.; at 11.44 A.M. 75 c.c. of air was

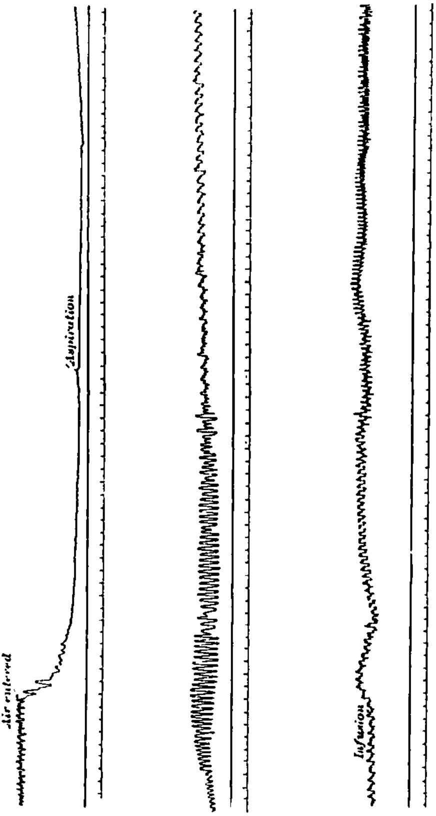
injected into the jugular vein. The blood-pressure immediately fell from 162 mm. to 50 mm.; the heart continued to beat perceptibly, the tracing on the drum showed less expansion, and the pulse was rapid, weak, and irregular. We did not resort to any treatment at this time. After waiting four minutes the pulse began to improve, and the pressure slowly rose until it reached 160 mm., and the dog was in quite as good condition as before the air was forced into the veins; thus in this case 75 c.c. of air had but a transitory effect on the heart. After waiting for twenty minutes (and during that time the heart remained in good condition) we forced 75 c.c. of air into the jugular vein (see Fig. 3); instantly the pressure fell, the femoral pulse disappeared, and the tracing on the drum was barely perceptible; in about two minutes the gasping respiration peculiar to these cases commenced. We then injected 50 c.c. of air; in three minutes after the first injection of air the dog was apparently dead. At this juncture the needle was inserted and the right ventricle was aspirated; in a few seconds a faint impulse was felt against the needle, and this was followed by a feeble tracing on the drum, and respirations began again. An infusion of normal salt solution at a temperature of 120° F. was begun, and the pressure slowly rose until in three minutes it had reached 180 mm., about a pint of salt solution in all having been used. After waiting twenty minutes the dog was killed with chloroform. On opening the chest no air was found in the heart or coronary vessels and no effusion of blood in the pericardium.

Experiment 15.—A fairly large dog weighing thirty-eight pounds was given 9 cgm. of morphine; at 10.30 A.M. anæsthesia with ether was started, and the operation was proceeded with as in the other experiments. At 11.08 A.M. the kymograph was started and a normal tracing obtained; at 11.10 A.M. 50 c.c. of air was injected into the jugular, and this was followed by a slight fall in blood-pressure, which, however, was speedily recovered from, and no treatment was necessary. At 11.15 A.M. 100 c.c. of air was injected; the pressure fell directly from 150 mm. to 12 mm.; no heart-beat was registered; no pulse could be felt; there were several gasping inspirations, and the dog seemed quite dead. After waiting some seconds to make sure that the animal would not recover spontaneously, and still no tracing showing any movement of the heart, the right ventricle was accordingly aspirated, and when a few ounces of mixed air and blood had been drawn off the heart's action could be felt against the needle, and also the tracing and pulse reappeared. In this case no infusion was done; the pressure rose to 150 mm., and the heart's action continued fairly good—a little irregular for a few minutes, and later quite normal. At 11.30 A.M. 100 c.c. of air was injected, causing a fall in blood-pressure, but the heart continued. (See Fig. 4.) At 11.35 A.M. the blood-pressure had risen to 120 mm., and 50 c.c. of additional air was injected. This was sufficient; the pressure fell, and there was neither pulse nor tracing in forty-six seconds. The dog being practically dead, the heart was aspirated, when the pressure rose to 72 mm. in one minute, the pulse being then slightly irregular and feeble. It continued in this manner until 11.40 A.M., when an infusion was done. This was followed by another rise in pressure to 125 mm., an increase in the force of the heart, and a regular pulse. The animal was finally killed by an injection of 100 c.c. of air into the jugular vein.

An autopsy showed the right ventricle filled with air; the coronary vessels contained air emboli; there was no effusion into the pericardium.

Experiment 17 (No. 1).—In this case we determined to do a sterile operation, inject air, aspirate only, and allow the dog to recover if he would. The dog in this case was a large, powerful bulldog, weighing forty pounds. He was accordingly given 1 dgm. of morphine at 10.30 A.M., and at 11 A.M. ether anæsthesia was started. The right carotid was opened, a manometer tube was inserted, and also the left jugular

FIG. 4.



vein was opened. No tracheotomy was done in this case. At 11.34 A.M. 125 c.c. of air was injected into the jugular. (See Fig. 5.) The pressure fell immediately from 140 mm. to 20 mm.; the heart's action ceased, no pulse being perceptible, and no pulse tracing was observed for thirty-two seconds; a needle was then introduced into the right ventricle and 150 c.c. of mixed air and blood withdrawn. Almost immediately the pressure began to rise and the heart's action to be

FIG. 5.

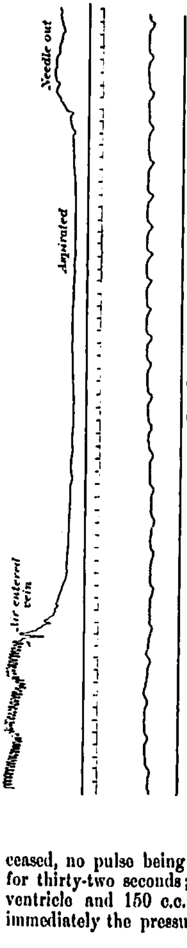
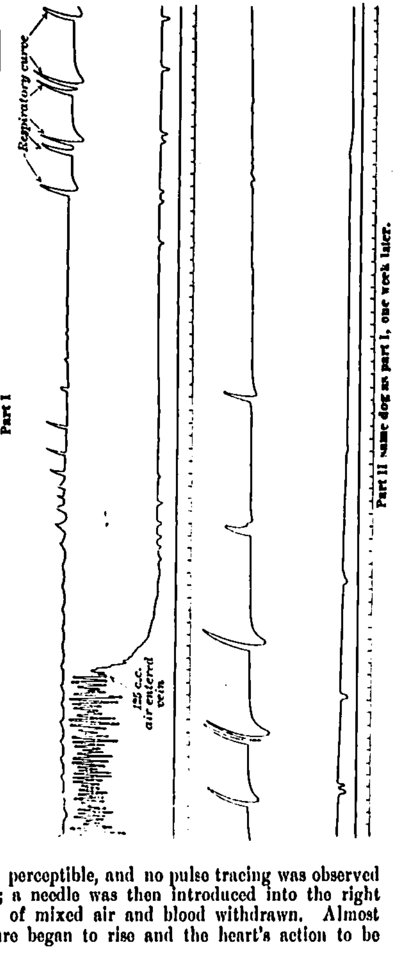


FIG. 6.



shown on the tracing. In this case the pressure rose very slowly, requiring one-half hour to reach 90 mm., and the expansion of the ventricles was greatly restricted compared with the normal tracing at first obtained. The wounds were sutured and collodion dressing was applied. The dog was placed in a cage and was surrounded by hot-water bottles. He recovered from the anæsthetic about 2 P.M.; when seen at 4 P.M. he was in good condition. The next day he was placed on a "regular diet." The dog recovered strength so rapidly that it was decided to repeat the experiment on the same animal.

Experiment 17 (No. 2).—On the sixth day the dog was given 1 dgm. of morphine, and later was anæsthetized; the opposite vessels to those used in the first case were then exposed and treated as before. In the second operation the respiratory tracing was attached to the kymograph to show the effects of air on the respiration more clearly. When the kymograph was started the large excursion in the heart tracing often noticed after morphine has been given was seen in this case. The heart's action was good; pressure 180 mm.—higher than it had been before the first operation. At 10.55 A.M. the respirations were 20 per minute; pulse 98. At 11.08 A.M. 125 c.c. of air was introduced into the jugular vein; immediately the pressure fell from 180 mm. to 28 mm., and in twenty-four seconds no tracing was perceptible on the drum and no pulse could be felt. There was little change in the respirations for several seconds after this, and they continued for two and one-half minutes after the heart had ceased, deep and gasping, as was beautifully shown on the tracing. (See Fig. 6.) The dog died, and on autopsy the right ventricle, as in other cases, was found dilated with air, and air was found in the coronary vessels.

The great excursion of the respiratory tracing in the preceding experiment demonstrates the deep inspiratory efforts of the animal, caused by anæmia of the brain centres when the cardiac circulation is interfered with. I think that this chart (Fig. 6) clearly shows—Hare's opinion to the contrary notwithstanding—that in the venous injection of air failure of respiration is dependent on cardiac failure.

In eleven experiments (from 7 to 17 inclusive), of which four have been described (*vide supra*), nine dogs recovered after aspiration and infusion, one dog died because the needle used was too small to aspirate the heart, and one dog died in spite of treatment. Two of the animals which finally recovered required large quantities of air (250 to 400 c.c.), injected forcibly, before the pulse tracings on the kymograph entirely disappeared. Three of the dogs which recovered were eventually killed by venous injection of air (see Experiments 10, No. 2, 15, and 17, No. 2), and on autopsy the coronary arteries were found injected with air, the right ventricle was dilated, and the heart was fibrillating. The introduction of the needle into the heart had no ill effects on that organ, and in but one case was any leakage into the pericardium found.

In applying the results of these experiments to the operating-room and the human subject I would divide the treatment into two parts—preventive and radical.

1. *Preventive.* When operating in the region known as the "danger zone" none but the prone position should be permitted, for should one of the veins be injured the semiprone or sitting posture predisposes to entrance of air into the veins. If any dissection is necessary, as is often the case, it should invariably be performed with a blunt instrument, never with a scalpel. Especial care should be used if the operation be one for the removal of cancerous or tuberculous glands, which are always more or less intimately connected with structures adjacent.

2. *Radical.* Should attention be called by the hissing sound accompanying the rush of air into the veins to the injury of a vessel in this region, the finger should be forced into the wound to prevent further entrance of air, the wound cavity should be filled with salt solution, and the chest should be compressed forcibly. If bubbles are seen in the fluid contained in the wound they are due to air coming out. If, however, the heart becomes tumultuous, if the churning sound attracts attention, and if the respirations become labored and the inspirations forced, a needle should be inserted into the fourth left interspace one inch from the left border of the sternum; it should be directed obliquely upward and backward, and the right ventricle will be entered; aspiration should be continued until the blood comes out unmixed with air; then, an assistant having previously prepared a solution of normal salt at a temperature of 115° to 120° F., the patient should be infused until fluid has been introduced equal in quantity to the blood taken from the heart. The most convenient region in which to perform this infusion is the median basilic vein.

To sum up, the following facts seem clear:

1. That entrance of air into the veins, even in small amounts, is to be dreaded, as it may result in death.

2. That death is due to gaseous distention of the right heart or to air emboli in the coronary vessels, and not to primary respiratory paralysis.

3. That combined treatment by aspiration and infusion we may expect to be attended with good results.

In conclusion, I would say that I believe the statement "that large quantities of air may be introduced into the veins without unfavorable result" to be pernicious teaching and not supported by fact.

[I wish to avail myself of this opportunity for thanking Mr. J. T. Hoyt for the very valuable assistance rendered me in his management of the apparatus during the course of these experiments.]

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A CASE OF SUBPECTORAL ABSCESS.

BY GWILYM G. DAVIS, M.D.,
OF PHILADELPHIA.

IN the issue of THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES for November, 1900, Dr. John H. Musser has gone over the literature of this affection, with an abstract of twenty-three cases besides those of his own.

The present case occurred in a man, aged thirty years. He was a worker in the boiler-room of a paper mill. Ten days previous to admission to the Episcopal Hospital, while firing boilers, he became overheated and went outside to cool off. He says this caused him to catch cold, and in a few days he noticed a lump forming on the left side of his chest. He stated that he was subject to colds and attacks of sweating. He was a Pole and spoke no English, and it was difficult to elicit the history.

On admission his temperature was 102° F.; pulse, 118; respiration, 28. On the left side of the chest was a large mass which extended from the nipple below to the clavicle above, and from the edge of the sternum out nearly to the deltoid pectoral groove. It was not red, but was tender and pitted on pressure, and deep fluctuation was plainly evident. The swelling was not pointed but flat, showing that the pus was deep seated. There was no tendency shown anywhere on the surface of pointing or breaking through. The axilla was absolutely healthy, and, as can be seen from the accompanying illustration, the anterior edge of the axillary fold was not encroached on. The depression which normally exists between the inner edges of the deltoid muscle and upper edge of the pectoralis major was perfectly preserved. There were no lesions on the left arm, but on the left hand there were the remains of several old cuts, but nothing recent. He failed to recall any injury to the affected parts or any recent suppurative lesions of the hand. He had an irritative cough with a scanty expectoration of white, frothy sputum. Physical examination of the chest was negative.